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Lameness and Poor Thriving in Lambs on Farms in Old Lead Mining Areas in the Pennines—I. Field Investigations

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History

EARLY in 1949 reports of a disorder among young Swaledale lambs, characterised by a specific locomotor disability which in the majority of cases resolves completely after a few weeks, were received from farms in the Alston Moor and Arkengarthdale areas of the Pennine district. Recovered lambs are stunted in growth and develop slowly, but apart from this the illness appears to leave no permanent disability as the females are kept for stock ewes and normally live for five, six or more years. The condition is apparently confined to young lambs two to ten weeks old and occurs on "affected" farms each year in May, June, and July, especially in backward seasons. Investigation showed that the condition is limited to areas which have been extensively mined for lead and it has not been observed elsewhere. All the affected farms are defaced with very obvious "dead heaps" or lead "raikes" which represent the debris from old lead workings and which contain a variety of lead ores. It is noticeable that herbage grows very sparsely or not at all on these heaps.

Evidence from a geological report for the Alston-Weardale-Arkengarthdale areas indicates that the rocks belong to the carboniferous limestone series, and consist of alternations of sandstones and shales, together with a few thick limestones having a more or less horizontal disposition. These rocks are traversed by a number of thin vertical walls or veins, carrying various minerals, among which is galena or lead sulphide. In the zone of circulating meteoric waters, which is above the permanent water table, and—except in the valley bottoms—lying some distance below the land surface, the galena has been altered to cerussite and anglesite, the carbonate and sulphate of lead respectively. Quantities of these minerals are present in the numerous refuse heaps or "raikes" resulting from extensive lead mining operations which have been carried out since the 16th century, and are consequently available to be licked by grazing animals. The system of working the lead veins by "hushing" or flood-washing has spread out these minerals in alluvial fans over the ground.

Marked symptoms of the disorder have been observed on six farms but it is probable that it occurs on many more farms in the areas concerned. It is a well-established belief that farms with lead "raikes" are prone to lamb stiffness which occurs each year in greater or lesser degree and although farmers believe that lead is responsible for the disability, the problem does not appear to have been investigated. As previously stated, the majority of affected lambs recover spontaneously, but in the autumn are of less value, as they never quite catch up on the stunting effects of the illness which may last in an acute form for two to four weeks. Cattle and calves are not affected, but in Arkengarthdale there is a belief that "lead" pastures are dangerous for horses as they may acquire a respiratory disorder known locally as "belland" which is attributed to chronic lead poisoning.

Symptoms

Stiffness of gait is the first noticeable symptom and may appear shortly after lambs begin to eat grass. Affected lambs cease to play, follow the ewes with a dejected air, and locomotion is slow, stilted and

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painful. There is no paralysis at this stage, but the flexor and extensor reactions are sluggish, the hocks and fetlocks are underflexed so that the feet tend to drag along the ground. In some seasons half or more of the lamb flock may show these symptoms, but in others about 10 per cent. of the lambs are affected. From this stage, and especially if their pasture is changed, most lambs tend to recover, but a proportion worsen to the stage of recumbency. The recumbent lamb often shows a well-marked paresis of the extensor muscles of the hind limbs and flexion of the fetlocks. This is shown in Fig. 1. There is partial or total loss of motor power, and sensory perception is inhibited; the fore-limbs may also be affected. When the condition has reached this stage it is probable that resolution cannot take place, but if well attended by their dams many recumbent lambs have been observed to recover. In cold weather, however, a number of these lambs succumb to intercurrent disorders. Although cases of swayback have been observed in these areas, the "lead lameness" is easily distinguished from swayback clinically by absence of inco-ordinated gait, and histologically by absence of demyelination of the central nervous system.

In 1949 several sudden deaths among two- to three-week-old healthy-looking lambs on affected farms provided an opportunity for preliminary *post-mortem* examinations. There was no evidence of dysentery or pulpy kidney disease but some of the kidneys were enlarged and their cut surface presented a well-marked, dark haemorrhagic band stretching in an arc around the boundary zone. No other abnormality was noted. Estimation of lead on liver and kidney tissues from two carcases showed values greatly in excess of the normal 1 or 2 p.p.m. It was therefore decided to investigate the possibility of chronic lead poisoning being the cause of the disorder. It is of interest that the lambs which died suddenly had not been observed to be ill or stiff and they provide an indication that the toxic hazard on these farms may be manifested in several ways, *i.e.* from a mild lameness ending in resolution, to an acute and rapidly fatal condition. Occasionally stiffness in aged ewes in the Alston area has been noted. This is ascribed locally to chronic lead poisoning but so far there is insufficient evidence on which to make a diagnosis. Since, however, it is chiefly young lambs that are affected, cumulative lead poisoning does not appear to be a common hazard among the adult sheep.

FIG. 1.—A severely affected lamb with paresis of the hind limbs and flexion of the hind fetlocks.

Investigational Procedure

During 1949, samples of blood from affected lambs and their dams, and samples of liver and kidney from any lambs that died or were killed, were taken for estimation of lead and copper; haemoglobin values were estimated on a few blood samples. The following winter and throughout 1950, groups of ewes and their lambs were selected from flocks on two affected farms and bled at intervals for estimation of blood lead values; samples of milk, faeces and herbage were also taken for estimation of lead.

On one large farm where it was stated that lameness in lambs occurred on one section of the farm (Flinty Fell) but was unknown on another section (Priorsdale Fell) live-weight gains of groups of lambs born on both these sections were followed by weighing them at weekly intervals from birth until nine weeks of age. For comparision, weight gains were also measured of a number of lambs on a hill farm (Otterburn) where the condition was unknown.

Lead was estimated by the method described by Blaxter & Allcroft (1950) and copper by a modification of the method of Eden & Green (1940).

Results

(*a*) *Blood Samples*

Lead, copper and haemoglobin values for blood samples taken during the summer and autumn of 1949 from affected lambs and their clinically normal dams were given in Table I (overleaf).

Blood lead values for " normal " sheep (*i.e.* sheep which have not had access to abnormal quantities of lead) have been found to fall within the range of 0.05 to 0.25 (mean 0.139) mg. per litre (Allcroft, 1950. The data in Table I, show that values for affected lambs are very much higher than this and indeed, the high figure of 2.50 mg. per litre is higher than the maximum found by Blaxter (1950) in a sheep dosed with 30 g. lead as the acetate. Even the values for the clinically normal ewes from "affected" farms are abnormally high.

The variation in blood copper values is similar to that reported by Eden (1941) for normal Northumbrian sheep and although a few low values were found there was no evidence of a general hypocupraemia such as is encountered in flocks where swayback in lambs occurs (Eden, Hunter & Green, 1945).

The haemoglobin values fall within the common normal range reported by Allcroft (1941). In a few cases serum calcium and magnesium and blood in-

TABLE I
LEAD, COPPER AND HAEMOGLOBIN VALUES FOR BLOOD SAMPLES FROM AFFECTED LAMBS AND THEIR DAMS

Reference	Number of animals	Pb mg. per litre		Cu mg. per 100 ml.		Hb g. per 100 ml.	
		Mean	Range	Mean	Range	Mean	Range
Lambs (clinically affected) ...	14	1·44	0·54-2·50	0·074	0·03–0·13	15·9	13·8–17·0
Ewes (clinically normal) ...	13	0·55	0·34–0·72	0·072	0·05–0·10	12·2	9·6–15·8

organic phosphorus was estimated. No abnormalities in these constituents were found.

In order to check blood lead levels of ewes during the gestation period, blood samples were taken from a group of ten stock ewes on two affected farms during the winter of 1949-50, one group being bled twice—at the beginning and again towards the end of the period. February values for ewes on one farm ranged from 0.20 to 0.34 with a mean of 0.230 mg. Pb per litre blood, while values for October and February for ewes on the other farm ranged from 0.28 to 0.48 (mean 0.364) and 0.18 to 0.48 (mean 0.352) mg. per litre respectively. The mean values for ewes on the former farm are only a little higher than those found in normal sheep as quoted above, while those for the latter farm are only slightly in excess of values found for 40 ewes which had grazed for one to six years on farms in Derbyshire on which old lead raikes were present (Allcroft & Blaxter, 1950). No symptoms similar to the condition now reported have been observed in lambs from these Derbyshire ewes nor from other farms in the Derbyshire area in which there are old lead workings. The mean blood lead content of the 40 Derbyshire ewes was 0.200 ± 0.010 mg. per litre compared with the normal lead content of 0.139 mg. per litre of sheep blood from non-plumbiferous areas.

Placental transference of lead in sheep has been demonstrated (Allcroft & Blaxter, 1950) but the mean blood lead levels of 0.230 and 0.364 mg. per litre found in the ewes on the two affected farms did not seem to be high enough to cause storage of harmful amounts of lead in the tissues of their foetuses. It was therefore decided to take blood samples at intervals after parturition from a group of ewes and their lambs from two affected farms A and G in order to see if there was any build-up in blood lead concentration after birth. A group of six ewes and their lambs on farm A were selected at random and marked for identification and on farm G a group of four ewes and lambs were similarly selected; blood samples were taken soon after parturition in April and subsequently at weekly intervals. Mean blood lead concentrations are shown graphically in Fig. 2. These results show, as would be expected, that blood levels for lambs at birth were very similar to those of their mothers, but soon afterwards they rose quite rapidly and within three to five weeks were double that of the ewe. This build-up was probably general in lambs on affected farms and could perhaps account for, or be a contributory factor to, the poor thriving of lambs on these farms. It is of interest, however, that in spite of these high blood lead concentrations, all but two of the selected lambs remained healthy and free from detectable lameness. The two affected lambs which were on farm A had blood lead values of 1.36 and 1.39 mg. per litre at the fourth week when symptoms of lameness were first detected. Another lamb on farm G, however, had a value as high as 1.69 mg. per litre at the same time but showed no abnormal symptoms. The results also show that there was an increase in blood lead concentration in the ewes during May and higher values were found at this time than had been found during the gestation period earlier in the year. The values for both lambs and ewes, however, are in line with values in Table I found for the clinically affected lambs and the clinically normal ewes which were examined the previous year.

(b) Milk Samples from Ewes

Towards the end of May milk samples were taken from the same group of ewes to see if high concentrations of lead were excreted by this route and thus be a major contributing factor in the accumulation of lead in the lamb. The lead content of the milk from five ewes was 0.13, 0.10, 0.07, 0.09 and 0.11 mg. per litre respectively. Blaxter (1950) reported values of 0.11 to 0.15 mg. per litre for normal ewes and since none of the values found for the ewes on this

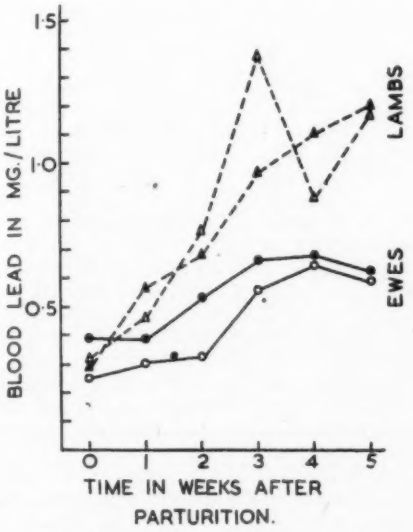

FIG. 2.—Mean concentration of lead in blood from groups of ewes and their lambs from two "affected" farms. Values for animals from farm A are represented by solid circles and triangles; those from farm G by open circles and triangles.

affected farm were higher, it does not appear that the milk of the ewes was one of the main sources of lead in the lamb.

(c) *Herbage and Faeces Samples*

Although the blood lead content of the lambs on affected farms was only slightly higher than normal at birth, it increased quite sharply over the first few weeks of life as shown in Fig. 2. Since symptoms of the disorder do not appear under two weeks of age while the lamb is entirely milk fed, it appeared probable that intake and absorption of lead increased considerably as soon as they began to eat grass. Samples of herbage were therefore taken during the summer from various parts of two affected farms for estimation of lead content.

TABLE II

LEAD CONTENT OF HERBAGE FROM AFFECTED FARMS, JUNE, 1950

Description of sample	Number of fields	Mean Pb content mg. per kg. dry matter	Highest Pb content mg. per kg. dry matter
Herbage from fells on which stiffness is prevalent	5	427	914
Herbage growing on or around "dead heaps" (*i.e.*, waste matter from lead mining operations) and old bore holes ...	4	261	480
Herbage from fells stated to be free of stiffness...	2	33	35
Healthy pasture from meadows around farm house	2	17	19

Results which are given in Table II show that herbage from areas in which stiffness occurs has a very high concentration of lead when compared with the usual 3 to 7 mg. per kg. on dry matter found for pasture samples from areas where soil does not contain much lead (Allcroft & Blaxter, 1950). The data also show a striking difference in the lead content of herbage on affected farms from the fells on which lameness occurred and from meadows said to be free of stiffness. The values for herbage from "affected" fells are also very much higher than values in herbage from Derbyshire taken from farms in the vicinity of old lead mining areas where concentrations ranging from 30 to 94 mg. per kg. dry matter were found (Allcroft & Blaxter, 1950). The high concentrations of lead found in these herbage samples are probably due to contamination by dust from the plumbiferous soil and rock in these old lead mining areas since it is known that the lead concentration of the leaf is not readily increased due to the high fixing power of soil (Keaton, 1937). Since the lead content of the herbage would depend on degree of soil contamination at the time the samples were taken, the wide variation reported in Table II is not unexpected. The sheep is, however, a selective grazer and a more accurate indication of the amount consumed would be obtained from faeces samples from sheep grazing over affected areas. Faeces samples were therefore collected during June 1950, from the rectum of some of the ewes and lambs comprising the two groups from which blood samples had been taken at intervals for estimation of lead.

TABLE III

LEAD CONTENT OF FAECES FROM EWES AND LAMBS GRAZING ON AN "AFFECTED" FARM, JUNE, 1950

Ewes	Pb in mg. per kg. dry matter	Lambs	Pb in mg. per kg. dry matter
E.1	1,180	L.1	1,000
E.2	—	L.2	206
E.3	134	L.3	517
E.4	80	L.4	87
E.5	1,170	L.5	86
E.6	123	L.6	183

It is evident from the data given in Table III that quantities of lead considerably in excess of normal were ingested by both ewes and lambs. Faecal lead values for sheep grazing non-plumbiferous areas have been found to range from 4 to 38 mg. per kg. dry matter (unpublished data) while those reported by Allcroft & Blaxter (1950) for ewes grazing areas in Derbyshire where the lead content of the soil is high, ranged from 28 to 275 mg. per kg. dry matter. Blaxter (1950) demonstrated that the apparent absorption of lead in sheep is very small and nearly all the lead ingested appears in the faeces. If it is assumed that the digestability of the dry matter of the grazing was 50 per cent., then dividing the values for faecal samples given in Table III by two, will give an approximation to the lead content actually ingested. This would give a range of 40 to 590 mg. per kg. dry matter which is in good agreement with the range of lead values of the herbage samples taken at the same time of the year.

(d) *Tissues from Lambs and Ewes on Affected Farms*

Samples of liver, kidney, and in a few cases blood and bones, were taken for estimation of lead from lambs and ewes which died on the two affected farms. The ewes and some of the lambs were the "normal" casualties that occur on hill farms, while other lambs died or were killed because they were severely affected with lameness.

The data in Table IV show that the lead content of all the tissues examined was much higher in affected lambs than in normal casualties. Indeed, many values found in tissues of lame lambs are as high as those found in tissues of acute lead poisoning in calves (Allcroft, 1950) but no symptoms of acute poisoning were observed in these lambs. It is obvious, too, that not all lambs on affected farms had an excessively high lead content of tissues, nor did any of the ewes which were found dead on these farms. Apart from the newly-born lambs, the lead concentration of tissues from normal lamb casualties are only a little higher than the 1 to 3 p.p.m. which is accepted as a common normal range found in grazing animals not exposed to abnormally high intakes of lead, and in most cases, they are well below the values found for lambs showing clinical symptoms.

Comparison of Weight Increases of Lambs on Affected and Healthy Areas

On one large farm it was stated that lameness in lambs occurred on one section of the farm but was unknown on another section. These sections were separated by natural boundaries and each carried its own separate sheep stock. The affected section—Flinty Fell—was lower in altitude and included much old meadowland which should have provided better pasturage than the healthy section—Priorsdale Fell, but the Priorsdale lambs had the better reputation for thriftiness, as well as freedom from lameness. Flinty Fell had many lead raikes whereas Priorsdale had very few, and it appeared possible that a higher lead intake might result in a depression in growth rate even though it might not be high enough to cause clinical symptoms. Blood lead values for a group of 10 healthy lambs from Priorsdale Fell ranged from 0.16 to 0.51 with a mean of 0.35 mg. per litre while a similar group from Flinty Fell had values ranging from 0.51 to 1.05 with a mean of 0.71 mg. per litre; values for affected lambs from Flinty Fell ranged from 0.96 to 1.27 with a mean of 1.12 mg. Pb per 100 ml.

An attempt was made to compare increase in weight of lambs from both these sections of the farm by weighing 20 normal-looking lambs from each at intervals from birth to nine weeks of age. On these extensive fells it proved impossible to collect and weigh all the 40 lambs each week but a sufficient number were weighed regularly to indicate weight increases in the two groups. These weights were compared with those for lambs of a similar breed on an excellent hill farm (Otterburn) in the same area where the condition did not occur.

The results indicated that the weight gains of the Priorsdale and Otterburn lambs were similar, both showing an average increase of 11 oz. per day over the nine-week period. The weight gain of healthy lambs from Flinty Fell averaged 8.5 oz. per day while affected lambs from this Fell averaged only 4.7 oz. per day.

This confirmed local opinion regarding the relative worth of Priorsdale and Flinty Fells and suggested the operation of some factor, other than general husbandry and nutrition, which had an inhibitory effect on growth and development of the Flinty lambs.

Discussion

The very high lead values found in tissues from affected lambs suggests that the lameness and poor thriving was a result of absorption of abnormal amounts of lead. The fact that tissue lead values for a few clinically normal casualties on "affected" farms were considerably lower than values found in lambs showing symptoms, suggests that not all the lambs absorbed abnormal quantities of lead even though they all grazed over similar areas. It is notable too that although faecal lead values of the group of ewes and lambs investigated (Table III) were of a similar range, blood lead values—and there-

TABLE IV
LEAD CONTENT OF TISSUES FROM LAMBS AND EWES ON TWO AFFECTED FARMS

Reference	Mg. per kg. fresh tissue		Mg. per litre	Mg. per kg. dry fat-free tissue	Remarks
	Liver	Kidney	Blood	Long-bone (Epiphysis)	
(a) Lambs showing clinical symptoms					
L.171	17·0	—	1·10	—	Four-week-old lame lambs. Recumbent, with loss of power in hind limbs. No other notable lesions detected on *post-mortem* examination.
L.204	30·0	16·5	2·70		
L.221	24·0	90·4	1·14	—	Six-week-old lame lamb. Showed complete loss of power in hind limbs with much fluid and bruising around hip joints. Destroyed. No other notable lesions detected.
L.380	30·8	143·0	0·97	—	Five-week-old lame lamb. Recumbent, no sensation in hind limbs.
L.389	13·4	6·9	1·24	316	Five-week-old lame lamb showing typical symptoms with well-marked extensor paresis and knuckling over at fetlocks. In poor condition but no other notable lesions.
L.397	22·5	5·4	—	184	Six- to eight-week-old lame lambs showing typical symptoms with well-marked extensor paresis. No other notable lesions.
L.398	11·0	13·4	—	—	
L.245	12·4	15·7	0·61	233	Six-week-old lamb showing typical symptoms of lameness with extensor paresis.
L.331	94·0	15·7	—	—	Well-developed three-week-old lambs found dead. No prior illness or lameness. Enlarged haemorrhagic kidneys in one.
L.332	47·0	39·1	—	—	
(b) Lambs and Ewes showing no symptoms, i.e., *"normal" casualties*					
L.259A	6·5	2·0	—	—	Seven- to ten-day-old lambs found dead. No specific disease diagnosed.
L.259B	7·8	2·4	—	—	
L.246	2·6	2·0	0·17	34	Week-old lamb affected with navel-ill.
L.259X	—	1·6	—	—	One-day-old lambs—found dead.
L.260A	—	1·6	—	—	
L.260B	—	2·4	—	—	
L.283	7·7	—	—	—	Three- to four-week-old lambs—no symptoms of lameness.
L.284	8·1	—	—	—	
E.154	—	4·8	—	—	Ewes found dead.
E.266	—	1·7	—	—	
E.170A	4·0	4·3	—	—	
E.170B	4·7	5·4	—	—	

fore absorption of lead—was always lower in the ewe than in the lamb. It is known that absorption of lead in sheep is very small and that nearly all the lead ingested appears in the faeces (Blaxter, 1950). Comparison of the lead content of herbage and faecal samples given in Tables II and III supports this finding. It has also been shown that lead in soil derived from weathered lead ore is less readily absorbed because of its lower rate of solubility and is, therefore, less toxic to calves than lead in the form of carbonate or acetate (Allcroft, 1950). The reason for the very much higher rate of absorption in some lambs than in others is not known. The few milk samples taken from ewes showed normal lead values and the evidence does not suggest that the lead concentration of the milk was sufficiently high to be harmful to the lamb. If it were harmful, one would expect to find similar concentrations of lead in tissues of all the lambs in a flock and a much higher proportion of clinically affected lambs. A possible explanation might be related to the fact that some lambs played and lay about on "dead" heaps on which there was no herbage. These lambs may have licked and ingested much greater amounts of soil high in lead ore than lambs which did not use these heaps, but no proof of this was obtained. On the other hand, lead may not be the only factor concerned in the aetiology of the condition. Although lead was the principal mineral extracted from these old workings, it is known that ores from many of them contained appreciable quantities of other metals as well.

The lead figures obtained for herbage samples indicate that a good deal of the herbage on affected farms was fairly heavily contaminated with plumbiferous soil dust, that ewes and lambs, therefore, must have ingested abnormal amounts of lead which did not always have harmful effects. Stock ewes are bred and remain on affected farms for years without showing symptoms of the disorder. A proportion of the lambs, however, became clinically affected and examination of tissues from them showed abnormally high concentrations of lead.

There is ample evidence in the literature (Cantarow & Trumper, 1944) showing that absorption of lead can cause a variety of toxic symptoms, and amounts only slightly in excess can, under some circumstances, result in detrimental effects on physiological functions. Although the evidence in this investigation is far from complete in incriminating lead as the sole cause of the disorder, there is no doubt that absorption of lead in affected lambs was abnormally high.

The possibility of another factor concerned in the aetiology cannot be ruled out and it was therefore decided to try to reproduce the disorder experimentally by dosing lambs with lead under both laboratory and field conditions. The results of this further investigation will be reported in a separate paper.

Summary

The history and symptoms of a disorder occurring among young Swaledale lambs in certain areas in the Pennines is described. The disorder is characterised by a specific locomotor disability and is confined to flocks grazing old lead mining areas.

Lead values for blood, tissues and faeces from affected lambs were abnormally high; herbage samples from affected areas also contained high concentrations of lead. This suggests that the condition was associated with absorption of abnormal amounts of lead although symptoms typical of acute lead poisoning were never observed and the majority of affected lambs recovered spontaneously; moreover breeding ewes on affected farms remained clinically healthy.

Blood lead concentrations of ewes and their newborn lambs were similar but within three to five weeks those of the lambs were double those of the ewes. Clinical symptoms were, however, not always associated with high blood lead levels.

A comparison of live-weight increases of groups of lambs from an "affected" and a healthy farm indicated that growth and development were considerably reduced in flocks grazing old lead mining areas.

Although these results suggest that absorption of abnormal amounts of lead are associated with the occurrence of the disorder, the possibility that other factors are also concerned cannot be ruled out.

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VETERINARY SURGEONS IN LITERATURE

Major R. C. G. Hancock writes:—

Many years ago E. V. Lucas wrote a delightful essay in the columns of the *Sunday Times* entitled "Vets" (*tout court!*). In this essay he stated: "We meet medical men by the thousand (in novels) but I cannot recall a single 'vet' outside the pages of Anstey's *Lyre and Lancet*."

Since then I have noted a few examples and have it in mind to compile an anthology of veterinary surgeons as depicted in our literature. Will anyone coming across such passages in novels, memoirs, magazines, and the like, let me know of their discovery and at the same time supply me with sufficient information to get to the source. Letters should be addressed to me at Meadham Cottage, Hannington, Basingstoke, Hants.

Preliminary Observations on Natural and Experimental Ringworm in Cattle

BY

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Introduction

IN recent years an increased interest has been taken in ringworm in animals, probably because it is being incriminated more and more frequently as a source of ringworm infection of man. Accurate figures of the incidence of cattle ringworm infection in man are not available, but during the last four years 30 cases have been referred to the Skin Department of the General Infirmary at Leeds because of their severity or their failure to respond to initial treatment. The preliminary studies recorded here were performed as part of a programme of research into ringworm in animals.

It is generally accepted that ringworm is common in cattle in this country and note was taken during the course of routine investigations in Yorkshire during the winter of 1952/53 of its incidence on the farms visited. In all, information was obtained from 43 farms ; the disease was present on 28 of these and in the majority calves in their first year of life were affected. The farms free of ringworm were dairy farms where calves were segregated at birth and reared in isolation, usually in hygienic surroundings. As a general rule, the standard of hygiene on affected farms was of a lower order ; many were beef or rearing herds where young stock had direct or indirect contact with older animals and were kept during winter in yards or buildings where woodwork and walls were seldom cleaned or painted. The incidence of the disease was not associated with any specific type of feeding, and no attempt was made to determine whether it was associated with any specific deficiency, although it was noted that the most severe and extensive lesions occurred in calves which were obviously undernourished. The disease was not confined to calves but was met in older cattle with a common history that the affected animals had not been infected during earlier life. Cultural examinations of hairs and scales were made from the lesions met with during the course of these observations and one species of ringworm fungus only, *Trichophyton verrucosum* Bodin var. *discoides*, Ainsworth & Georg, was isolated. This also applied to the human cases examined.

The survey was not continued for it was felt that any additional information so gained would add little to our present knowledge of the disease, and it was decided to concentrate on a study of the host-parasite relationship in natural and experimental ringworm infection of calves induced under controlled conditions.

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Methods

1. *Calves used for experimental infection* were kept in individual pens in a concrete building in which animals had never before been housed. There were three pens, 5 by 4 feet, on each side of a central passage 3 feet 6 inches wide. The partitions were concrete slabs 4 feet high and the doors were of wood, also 4 feet high. The calves were bedded on wheat straw and fed a ration of oats, flaked maize, calf nuts and good quality hay. Individual feeding and watering buckets and halters were used to avoid any chance spread of ringworm infection. Three naturally infected calves used as a source of infective material were housed in an old-fashioned stable with wooden walls and received the same food as the experimental calves. All experimental calves remained in excellent condition and maintained an even rate of growth.

2. *Preparation of Infecting Suspensions.* Epithelial scales and hairs from typical ringworm lesions in the donor calves were wetted and broken up in normal saline to form a uniform suspension immediately before experimental application. The presence of spores and mycelium in these suspensions was confirmed microscopically and culturally. Suspensions were also prepared from cultures by scraping colonies of *T. verrucosum* var. *discoides* from a solid medium and emulsifying them in saline.

3. *Microscopical and Cultural Methods.* For preliminary examination scales and hairs were mounted on a slide in 10 per cent. potassium hydroxide and examined microscopically after a few minutes. Mycelium could be demonstrated in the epithelial scales and mycelium and spores in the hairs. Permanent preparations were also made by scraping epithelial debris on to slides treated with albumen. These were stained by the Hotchkiss-McManus modification of the periodic acid-Schiff method (Fig. 1). Selected infected material was planted on Sabouraud preservation medium and honey peptone agar. In later examinations the cultural techniques described by Austwick (1954) were adopted.

4. *Histological Examinations.* Skin biopsy samples were fixed in 10 per cent. formol saline, care being taken both during the removal of the sample and the subsequent techniques to avoid as far as possible separating the matted exudate covering the site of infection from the underlying skin. Sections were stained with periodic acid-Schiff followed by haematoxylin. This method was found most useful for demonstrating not only the organisms but also the

tissue reaction. In addition to using this technique for the staining of many of the sections we combined the P.A.S. reaction with the staining technique used by Auber (1950) in order to obtain detailed information regarding the selective progress of the fungus in tissues undergoing keratinisation.

Results

In the preliminary experiments, sites were shaved on the withers and back of a female Ayrshire calf, approximately six months old. A day later two sites were prepared for inoculation by lightly scarifying with fine sandpaper to cause slight abrasion of the area but without exudation of blood or serum. Two other sites were prepared by a more severe scarification with a scalpel sufficient to cause an exudate. Saline suspensions of natural and cultural material were rubbed into those sites and also into unscarified sites. Slight reddening was first seen 20 days later on the sandpapered site inoculated with natural material, and this was well established 42 days after inoculation when spore sheaths were demonstrated in hairs removed from all the sites infected with natural material. At this stage the areas scarified by scalpel and inoculated with culture material were also positive microscopically. All sites were positive at 70 days.

None of the lesions showed the typical thick whitish yellow scaly crust with embedded hairs seen in the natural disease but were of a more inflammatory nature, depilated at the centre with crusted exudate round the edges. As some of the sites were on the back, the animal could lick them and it was decided that this accounted for their appearance. Fourteen weeks after the experimental inoculations and while spores and mycelium could be demonstrated on the edges of the lesions, new non-infected hairs had commenced growing in the centre. During the course of the experiments a lesion of normal appearance developed over the left eye, presumably acquired from the experimental sites.

Subsequent experiments in three other calves were performed and it was found that there was no need to roughen or scarify the skin, but if the hair was clipped short and the infective suspensions rubbed in lightly with a handle of a scalpel or with the finger, infection was established. It was also found that if sites on the neck which could not be licked by the animal were chosen, the experimental lesions bore a closer resemblance to those seen in the naturally acquired disease. Biopsy specimens were removed from these experimentally induced lesions at seven-day intervals in order to observe the initial infection process.

The experimentally induced lesions in these three calves followed a similar course. A disturbed hair pattern was first noticed closely followed by erythema at the site of inoculation between 14 and 21 days after infection and at this stage it was possible to demonstrate mycelium in epidermal scales and spores on the hairs. The typical lesion of ringworm with heavy greyish white plaques of crust in which broken off and growing hairs were embedded gradually developed within 30 days. When the crusts were removed, moist serous areas were seen, in which healthy hairs were beginning to grow. There was no scarring. Within a period of four months the experimental lesions healed spontaneously and no indication of infection could be detected clinically or microscopically.

Natural Infections

Two healthy Shorthorn male castrated calves were put into the stable which had previously housed the donor calves and which had not been cleaned out. These calves developed natural lesions within 28 days and biopsy specimens were removed at various periods during the later development and course of the lesions. It was not possible to detect these lesions until infection was well established so the biopsies were only of value in observing the lesion at its height of activity and during the subsequent healing process, which was complete after approximately three months.

Reinfection

Two calves which had recovered from experimental infection and in which the lesions could no longer be demonstrated were inoculated on fresh sites on the neck and croup with infective material; in one calf within two months of experimental infection, and in the second, five months after. In the first calf, scaling and slight reddening with some thickening of the skin was observed eight days after inoculation and mycelium was observed in scrapings from one lesion only. Within three days the reaction had disappeared and it was no longer possible to demonstrate the presence of fungus. Lesions developed in a normal calf inoculated at the same time with a portion of the same infective material. The second calf was inoculated on sites on the neck with natural and cultural material but there was no reaction and no mycelium could be demonstrated in preparations from the lesions.

Histopathology of Biopsies

The following features relating to the distribution of the parasitic fungus in ringworm lesions caused by *Trichophyton verrucosum* var. *discoides* were observed in serial sections from biopsy material.

In the early stages of invasion there was an extensive proliferation of mycelium in the stratum corneum. This included the stratum corneum which lined the infundibulum of the hair follicles (Figs. 2 and 3). In many of the follicles a thick weft of mycelium was formed around the hair shaft at the mouth of the follicle (Fig. 4). The mycelium then frequently penetrated into the intrafollicular stratum corneum, growing along its whole extent (Fig. 5). In a few sections it had even grown down into the inner epithelial sheath as far as the trichohyalin zone (Fig. 6). The sheath of spores which subsequently developed was formed by the transverse fragmentation of a mycelial network which in some cases appeared to originate in the mycelial weft at the mouth of the follicle, or in the mycelium in the intrafollicular stratum corneum (Fig. 7).

Downwardly branching hyphae were present on the surface of the hair shaft. In follicles in which a spore sheath was already present, hyphae had penetrated between the overlapping cuticle scales of the hair shaft and entered its cortex (Fig. 8). The mycelium was, in many cases, abundant in the cortex as parallel

FIG. 1.—Mycelium and spores in hair and scales in scraping. Experimental lesion. Stained P.A.S. × 200.

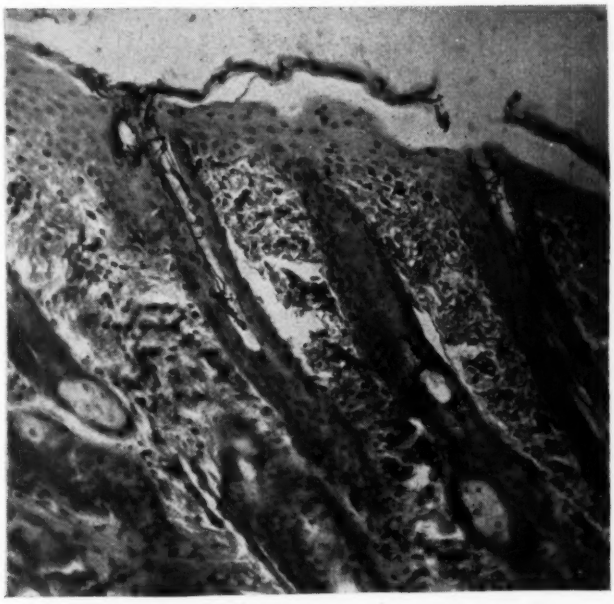

FIG. 2.—Mycelium in stratum corneum and in hair follicles. Experimental lesion. Stained P.A.S. and Haematoxylin. × 200.

FIG. 3.—Mycelium in stratum corneum which is infiltrated with inflammatory exudate. Experimental lesion. Stained P.A.S. and Sacpic.* × 514.

* The term sacpic refers to Auber's staining technique and is the term which he himself used in his text.

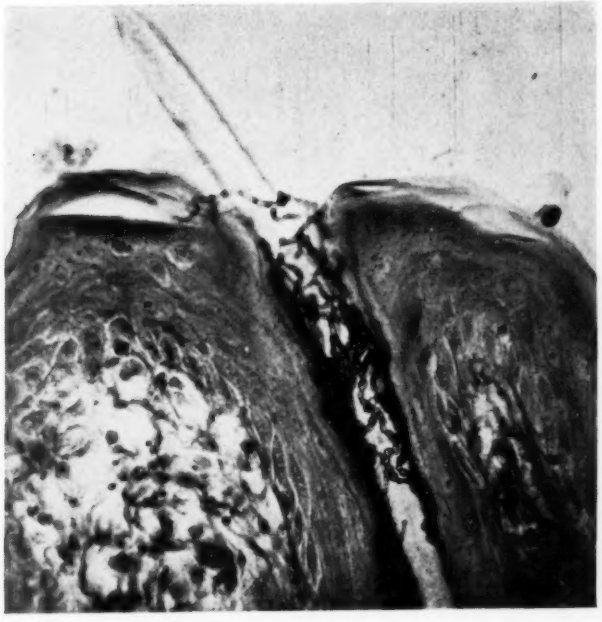

FIG. 4.—Mycelium surrounding hair shaft at upper end of follicle. Experimental lesion. Stained P.A.S. and Sacpic. × 514.

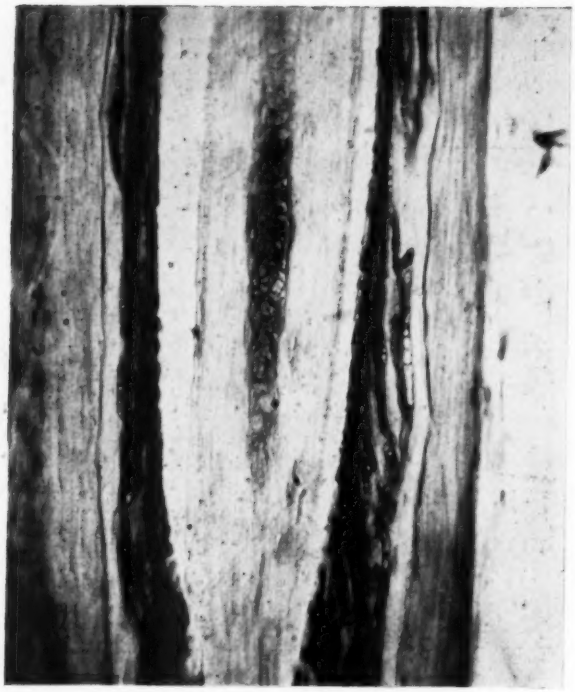

FIG. 5.—Mycelium in inner epithelial sheath of hair follicle. Experimental lesion. Stained P.A.S. and Sacpic. × 514.

FIG. 6.—Mycelium (m) in hair shaft and in inner epithelial sheath, reaching almost to trichohyalin zone (Tz). Experimental lesion. Stained P.A.S. and Sacpic. × 514

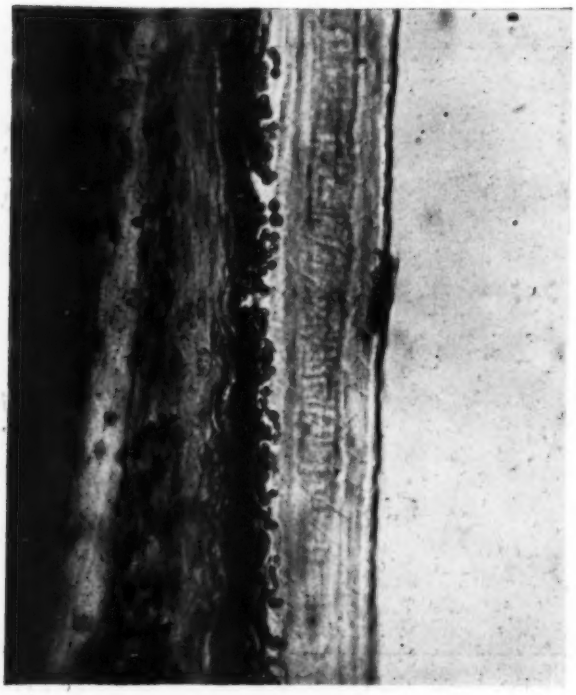

FIG. 7.—Mycelium in intrafollicular strateum corncum, forming spores. Naturally acquired lesion. Stained P.A.S. and Sacpic. × 514.

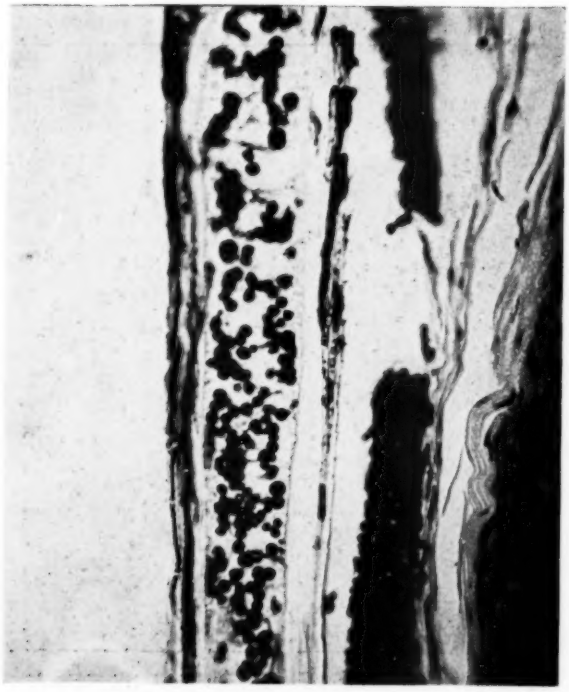

FIG. 8.—Spores and chain mycelium in medulla of hair shaft. Mycelium entering hair shaft between cuticle scales. Naturally acquired lesion. Stained P.A.S. and Haematoxylin. × 514.

FIG. 9.—Mycelium (m) forming Adamson's fringe around apex of keratogenous zone (k). Also mycelium in inner epithelial sheath. Naturally acquired lesion. Stained P.A.S. and Haematoxylin. × 514.

FIG. 10.—Bulb of "bed" hair invaded by mycelium. Experimental lesion. Stained P.A.S. and Sacpic. × 514.

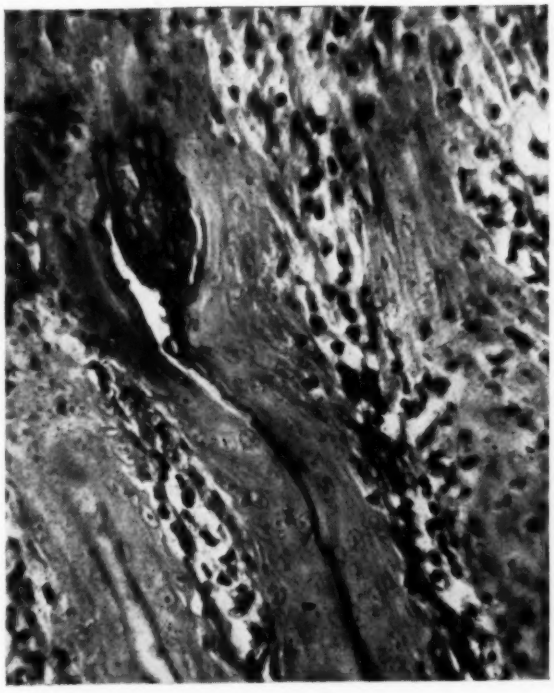

FIG. 11.—Mycelium entering pilosebaceous gland duct from hair follicle. Experimental lesion. Stained P.A.S. and Sacpic. × 514.

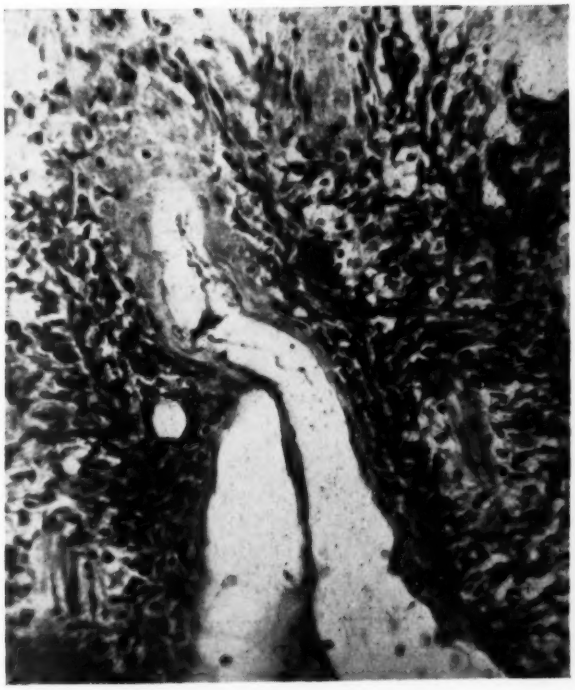

FIG. 12.—Mycelium in pilosebaceous gland duct. Experimental lesion. Stained P.A.S. and Sacpic. × 514.

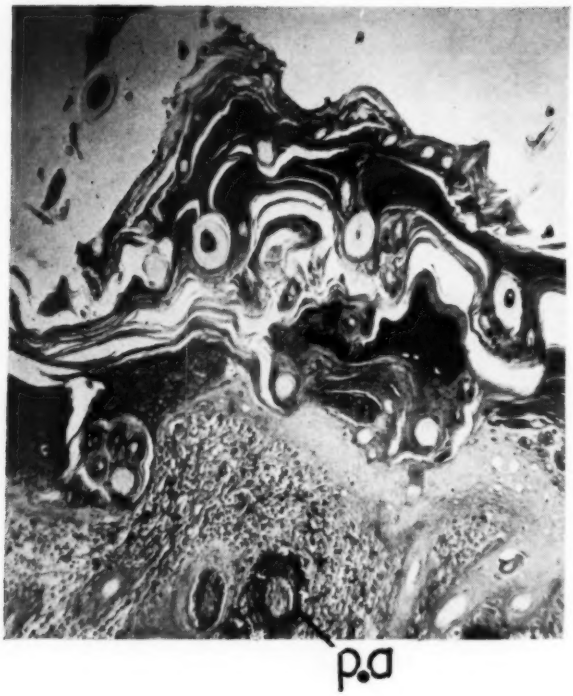

FIG. 13.—Inflammatory reaction to invasion of fungus. Infiltration and undermining of stratum corneum by inflammatory exudate. Infiltration of corium by inflammatory cells, and perifollicular abscess (p.a.). Experimental lesion. Stained P.A.S. and Sacpic. × 120.

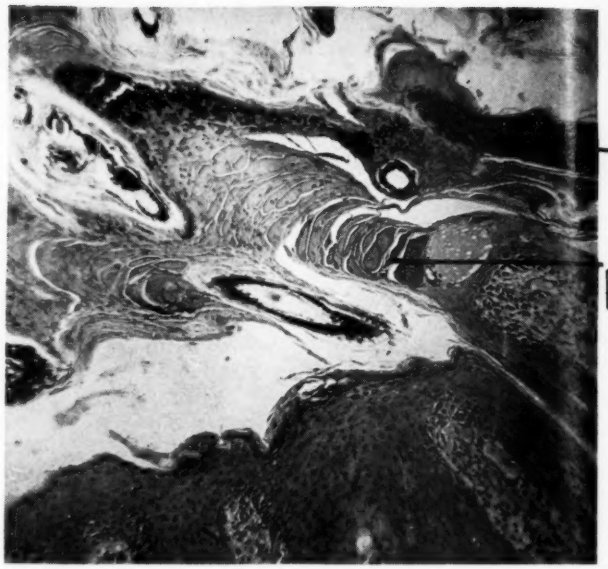

FIG. 14.—Inflammatory reaction to invasion of fungus : Cone of parakeratotic cells (p.c.) of stratum corneum distended with serum ; pools of pus (p.). Naturally acquired lesion. Stained P.A.S. and Haematoxylin. × 120.

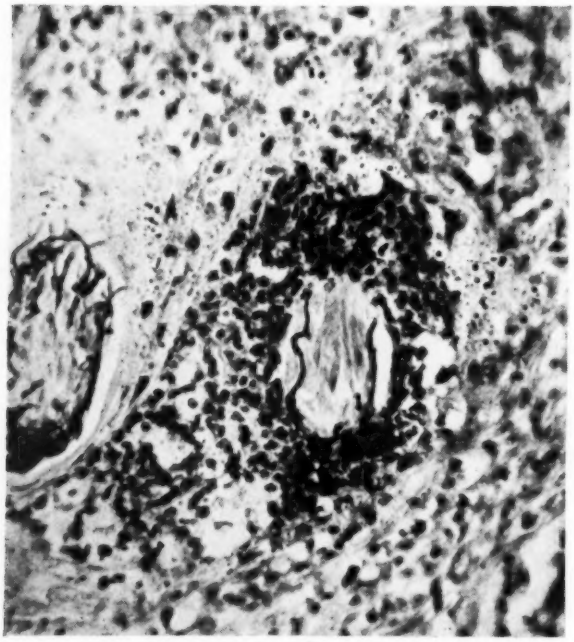

FIG. 15.—Perifollicular abscess around fragment of infected hair. Experimental lesion. Stained P.A.S. and Sacpic. × 120.

FIG. 16.—Infected hair becoming detached from follicle, and surrounded by sheath of hyperkeratosed stratum. Naturally acquired lesion. Stained P.A.S. and Sacpic. × 200.

downwardly branching hyphae, many of which were closely septate (chain mycelium). The tips, in many of the hair shafts, formed a fringe around the apex of the cone-shaped keratogenous zone which surmounts the bulb (Fig. 9). In some of the hair shafts, the medulla was filled with spores (Fig. 8). The keratinised bulbs of many shedding hairs (bed hairs) were surrounded and penetrated by mycelium (Fig. 10), although the shafts of such hairs did not appear to be penetrated to any great extent. Mycelium was present also in the ducts of some of the pilosebaceous glands (Figs. 11 and 12), occasionally as far as the gland itself.

Tissue reactions

Coinciding with the early clinical signs of infection there was a marked inflammatory response to the invasion of the parasite. This response was at first localised, but became in due course more widespread and was accompanied by an excessive proliferation of the stratum corneum into which inflammatory exudate had infiltrated (Fig. 13). Surmounting many of the papillae were stratified accumulations of parakeratotic horny cells dilated with serum (Fig. 14). In due course, intradermal and intra-epidermal micro-abscesses were formed, many of which surrounded infected follicles (Fig. 15). In some cases there were also accumulations of leucocytes within the lumen of the follicle. Many of the intra-epidermal micro-abscesses had ruptured beneath the stratum corneum undermining it extensively with pools of exudate containing large numbers of leucocytes. Some of the pilosebaceous glands were infiltrated by leucocytes. No giant cells were seen in any of the sections.

Discussion

The experiments described, although of a preliminary nature, have confirmed that cattle ringworm is a readily transmissible disease. During the course of the experiments accidental cross infections occurred on two occasions within the clean calf house ; one was clearly a contact case but in the second the possibility of airborne infection could not be ruled out. Also, healthy calves contracted ringworm in a stable which had previously housed infected calves. These observations, together with those made on the incidence of the disease in the field, have led us to conclude tentatively that the higher incidence of ringworm in the winter is due in the main to the concentration of animals in an infective environment.

Lesions in artificially and naturally infected calves healed spontaneously within four months of initial infection, and the healing processes were clearly seen in the histological preparations which gave evidence of inflammatory and reparative stages. It is a common experience that ringworm lesions disappear in the spring and early summer soon after infected animals are turned out to grass ; this might be due to changes in nutrition and environment but might possibly reflect the fact that the period between infection and turning out coincides with the natural course of the disease. Nevertheless, there is ample evidence that on many farms, calves, individually or collectively, develop extensive and spreading lesions which may cover large areas of the skin. Such infections are most commonly seen in calves on a low plane of nutrition as evidenced by stunted growth and poor condition. This type of ringworm occurred in calves suffering from an experimentally induced vitamin A deficiency, whereas control calves, kept under identical conditions of feeding and management, but receiving an adequate supply of vitamin A, recovered spontaneously. (Blakemore, Ottaway, Sellers, Eden & Moore, 1950).

These various factors must all be borne in mind when treating naturally occurring cattle ringworm and assessing the value of the various therapeutic agents which may be used.

The two reinfection experiments, although inconclusive in themselves, suggest that previously infected animals resist infection, as has been observed by Hoerlin (1945). In the field, ringworm infection is seldom seen in adult animals on farms where the disease is prevalent in calves, although these adults may be exposed continuously to infection.

It is clear from our histological examination of biopsy sections (including both naturally acquired and experimental lesions) that *T. verrucosum* var. *discoides* proliferates abundantly in the stratum corneum and that it penetrates deeply into the follicles and hair shafts. The general opinion is that dermatophyte fungi confine their parasitic activities to tissues in which keratin is found ; this is, by and large, substantiated by our observations. In view of this, the presence of the fungus in some of the ducts of the pilosebaceous glands is puzzling, for the mycelium is not confined to the keratinised lining of the ducts but is, in fact, more prominent in the lumen. The presence of spores in the medulla of the hair shaft is interesting. In our view it represents the final objective in the process of invasion of the hair by the parasite, for this refuge is a well protected reservoir for the spores. An apparently similar observation has been made by Birt & Wilt (1954) in the case of human hair parasitised by *T. verrucosum* and also by one of us (La Touche, 1953) for cat hair infected by *Microsporum canis Bodin*.

The mode of origin of the spore sheath is another feature of interest. Our present observations in this connexion seem to provide the explanation for the fact recorded by one of us in a previous communication (La Touche, 1952) that the spore sheath could be removed easily from hair shafts mounted in 10 per cent. caustic potash solution for microscopical examination, and that it was sometimes removed together with the epithelial lining of the follicle.

As regards the reaction of the host to infection, it is clear that the parasite provokes in the lesion a vigorous inflammatory response. This, in addition to being a defence mechanism, provides the natural means by which the parasite is eradicated from the lesion, for the inflammatory exudate, on hardening, forms a crust in which are embedded both infected hairs and scales. When this crust is removed by one means or another, the infective hairs are removed from the follicles, carrying with them most of the parasite (Fig. 16), and leaving behind a relatively healthy area.

Summary

1. Ringworm due to *T. verrucosum* var. *discoides* was reproduced in calves by inoculating material from natural lesions or culture suspensions on to prepared sites on the skin.

(*Concluded at foot of col. 1 overleaf*)

ACTH in the Treatment of Acute Induration of the Bovine Udder

BY

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THE treatment of the severely inflamed udder (usually a result of staphylococcal infection) has proved unsatisfactory owing to the very slow response to accepted methods. However, in the light of its application in the human field in cases of severe inflammation, the writer decided to use ACTH in a series of cases.

Durrell (1954), working in America, records the use of ACTH in a case of mastitis due to *B. coli* which had failed to respond to large and repeated doses of the whole range of antibiotics. The response was dramatic to a combination of ACTH with oxytetracycline and other antibiotics.

ACTH has been tried by the writer in some 20 cases, three of which failed to respond. Of these three, two were not true cases of mastitis in that the milk was unaffected. In both there was a localised inflammation of unknown aetiology with no systemic effect. The third case was that of an aged cow with a large "dropped" udder and staphylococcal mastitis in all four quarters. This case failed to respond to the whole range of antibiotics in combination with ACTH when applied both systemically and locally.

Of the 20 cases, 13 were immediately pre- or post-calving cases, clinically diagnosed as having developed mastitis of staphylococcal origin and showing systemic effects. Although routine samples were taken of untreated cases, the majority had been "tubed" before being seen. One case was of infection by *C. pyogenes*. It was estimated that all the affected cows would have been left, after antibiotic therapy, with an indurated quarter.

Previously, in such cases, we have tried the following modes of treatment: repeated massage; local and systemic antibiotic therapy; drainage of the duct system by intravenous posterior pituitary extract, followed by infusion of a large bulk of antibiotic solution; direct injection of antibiotics into the udder tissue.

By repeated local and general antibiotic therapy it is usually easy to obtain a return to normality of the udder secretion, but in very reduced quantity. Usually, however, the inflammation remains until replaced by fibrous tissue and the quarter is of little further use.

Direct injection does, on occasion, produce spectacular results, but the extra damage produced does not justify the routine use of the method.

In all cases except those specified above, response to the use of ACTH was good, in that the inflammation of the quarter largely disappeared within a few days of cessation of treatment. For obvious reasons the milk yield was reduced in the affected quarter; but it is hoped that the much earlier return of the quarter to normal will have its effect on subsequent yields.

Owing to economic factors 50 I.U. only were used in the early cases, but the response justified raising this to 100 I.U., followed by 50 I.U. 48 hours later. There seems little doubt that even higher doses would be still more effective if justified by the value of the animal.

In all cases, the treatment was combined with local and general antibiotic therapy; the dangers of suppressing an essentially defensive mechanism such as inflammation, without suppressing its cause, being obvious.

Discussion

The mechanism of the action of ACTH in inflammation is not yet clearly understood, nor have the relative importance of its direct effect and that via the adrenal cortex producing increased output of cortical steroids been evaluated. There are, however, several theories. Durrell attributes his response partly to indirect stimulation of the adrenal cortex producing an increase in the animal's resistance to stress; and partly to the known synergism between ACTH and many of the antibiotics.

Menkin (1954), in a long series of experiments, has shown that ACTH depresses capillary permeability in inflamed areas, by stimulation of the adrenal cortex and production of cortisone with its depressant effect on leucotoxin. He also has shown conclusively that ACTH has a direct effect by antagonising exudin and so greatly reducing fluid loss into the intercellular spaces of the inflamed area. This second effect persists even in the adrenalectomised animal. Menkin also suggests

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Preliminary Observations on Natural and Experimental Ringworm in Cattle—*concluded.*

2. Provided a site was chosen which could not be disturbed by the animal, experimental lesions more closely resembled the natural disease. Healing was spontaneous over a period of four months.

3. Biopsy specimens indicated that infection was a gradual process of invasion of the keratinised layers of skin and hair follicles.

4. The experimental findings are discussed in relation to field observations and it is suggested that concentration of animals in an infective environment, natural healing processes, dietary inadequacies and resistance (immunity) may all play a part in the epizootiology of the disease.

Acknowledgments.—We are grateful to the Agricultural Research Council for the grant which has enabled us to carry out this Research; to Dr. Ingram, Head of the Department of Dermatology, General Infirmary at Leeds, for laboratory facilities, advice and encouragement; to Dr. L. Auber, Wool Industries Research Association at Leeds, for kindly demonstrating his staining methods; to the Department of Clinical Photography for processing our photomicrographs; to Mr. R. Forster for his technical assistance especially in regard to the preparation of the histological sections and to Mr. R. Parker for his management of the calves.

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BLAKEMORE, F., OTTAWAY, C. W., SELLERS, K. C., EDEN, EVA, & MOORE, T. (1950). (Unpublished data.)
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that a further effect exists in the suppression of cell cleavage in areas of great activity which is exhibited both directly and indirectly by ACTH. In the same work he mentions unpublished evidence suggesting a suppressive effect on the formation of leucotoxin and the leucocytosis-promoting factor.

Michaud (1954) has described the effects of cortisone and hydrocortisone as tending to reduce hyperaemia, cellular exudation and infiltration and proliferative changes, pain and swelling in inflammation. These effects will be produced indirectly by ACTH.

Both ACTH and cortisone find application in human medicine in the treatment of severe inflammation, particularly of the skin ; but also in corneal ulceration where scar formation is prevented by suppression of inflammation and repair by fibrosis.

The failure of the drug in the two cases in which no infective agents were incriminated and no antibiotics used would seem to indicate that the synergistic effect of ACTH must play an important part in the response. This effect alone, however, would not account for the reduction of the inflammatory response but only for reduction of its cause ; the evaluation must await further research.

Previous methods have left the quarter or udder a distorted mass of fibrous tissue often containing sources of persisting infection which may flare up at a later date. It is hoped that this method will prevent such occurrence and also increase the likelihood of return to normal lactation.

During the " summer-mastitis " season it seems possible that the drug will be of use in the treatment of the acute induration produced in these cases. Though we have only had opportunity to use the method in one case of infection by *C. pyogenes*, response was obtained.

Finally, it must be stated that the method is not claimed as a spectacular advance, being neither dramatic nor infallible. It is merely hoped that it may be of use to others who, like the writer, have failed to solve this problem by other means.

Acknowledgments.—Thanks are due to Mr. D. W. Wood, B.SC., M.R.C.V.S., for permission to use, and encouragement during the trial of, the drug, and also to Miss Raymer, of the R.C.V.S. library, for provision of the literature.

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DELIVERY OF " THE RECORD "

We have received several letters from readers complaining that their copies of last week's issue were delayed. The reason was a temporary hitch in production beyond the control of our printers who, indeed, overcame the difficulty so speedily that the bulk of copies were posted to schedule.

Current Literature

ABSTRACT

Myopathy in Sheep. Its relationship to Scrapie and to Dermatomyositis and Muscular Dystrophy. BOSANQUET, F. D., DANIEL, P. M., & PARRY, H. B. (1956). *Lancet.* Oct. 13th, 1956, pp. 787–46. 21 figs.

This paper describes a hitherto unrecorded spontaneous degeneration of skeletal muscles (myopathy) in adult sheep, which in many ways resembles the diseases of dermatomyositis and muscular dystrophy in man. In the paper the term " myopathy " is used to mean a pathological degeneration of muscle of unknown cause. Dermatomyositis in man has been described as a polymyositis in which widespread changes in muscles do not show perivascular infiltration by inflammatory cells as a striking feature so that the changes in muscle are not a true inflammation but rather an acute degeneration of muscle. Pruritus is sometimes a feature of human dermatomyositis. In man muscular dystrophy differs from polymyositis (dermatomyositis) in often being inherited, in having several distinctive distributions of the muscles affected, and in its extremely chronic course over many years, although the more chronic forms of dermatomyositis without initial constitutional signs may not be unlike muscular dystrophy.

(It will be realised that the muscular dystrophy of man referred to in this paper is quite unlike muscular dystrophy of calves associated with deprivation of vitamin E.).

Most of the affected sheep providing the material on which this paper is based were suffering from scrapie, and the myopathy accounts for many of the clinical features of this disease. In addition the myopathy has been found in two other clinical syndromes, which have not been previously recorded. It is pointed out that scrapie and related myopathies are widespread in sheep in Britain and are of very considerable economic importance. This account is based on the study of 439 sheep affected with myopathy. Of these 45 were studied fully both clinically and pathologically. The necropsy included dissecting out all the skeletal muscles to determine the extent and severity of the myopathy. As controls 15 unaffected sheep were studied in the same way. The affected sheep came from flocks widely scattered over 12 counties in Britain. The sheep were mainly kept in folds on arable crops and were well fed especially during adolescence.

For recognition of the earliest signs of the myopathy the authors relied on the observations of experienced shepherds. As soon as the shepherd noticed signs of illness the sheep were brought into hospital for observation for periods varying from a few days to six months.

Some sheep died of their disease but most of them were killed, usually by a rapid injection of an overdose of sodium pentobarbitone into the jugular vein.

Details are given of the *post-mortem* technique and the methods used to preserve and fix tissues. There then follows a detailed description of the morbid anatomy of the tissues and a report of the histology

of the muscle changes and of the nervous system. The 21 figures deal with the macroscopic and microscopic changes in muscle.

The outstanding lesions were degeneration of the skeletal muscles, which may show itself as enlargement and extreme pallor of whole muscles ; as small foci of pallor widely disseminated ; as generalised wasting of skeletal muscle, with changes of texture in the muscles which come to resemble fish-flesh and are translucent ; or as an unusual firmness of the muscles without wasting. These various forms of muscle change may be found in the same animal, and the lesions are astonishingly widely scattered. They tend to be symmetrical, but occasionally the muscle on one side of the body is much more severely affected than that on the opposite side.

All the muscles examined from both normal and myopathic sheep have been infected with sarcocysts. There was no cellular response to the presence of the sarcocysts and the writers agree that there is no reason to implicate this universal parasite of sheep in the genesis of scrapie.

Examination of the nervous system showed no obvious loss of anterior-horn cells in the spinal cord such as would have been found in a motor-neurone disease. There were no inclusion bodies, no foci of nerve-cell degeneration and no perivascular cuffing which would indicate a virus infection of the central nervous system. "*The occasional vacuoles found in the large cells of the medulla and rarely in the anterior-horn cells of the spinal cord (which have aroused so much interest in all workers on scrapie) are, we believe, of little significance, because we have seen them in the nerve-cells of normal sheep and of humans affected by various diseases.*"

The skin lesions showed a non-specific increase of cells in the dermis but no obvious abnormality of the collagen. Neither the skin lesions nor organs other than muscle have as yet been the subject of full study.

There then follows a description of the principal clinical features presented by the sheep studied. It is pointed out that sheep affected with myopathy show a wide variety of clinical signs, which give rise to a bewildering number of syndromes, with many intermediate forms. According to the writers many of the animals undoubtedly had scrapie. They show a progressive loss of power of the skeletal musculature, usually with marked loss of muscle mass and a disturbance of gait and posture which can be closely correlated with the muscles most severely affected by the myopathy. Many animals rub themselves or scratch with their hind legs without signs of any skin irritation and some have a rash with a papular eruption of the haired skin.

The writers state that though scrapie is usually slowly progressive and fatal they suspect that occasional cases may become stationary and even regress.

The onset of the disease (myopathy) is extremely insidious. The earliest changes are disturbances of locomotion, disturbances of consciousness, and changes in the appearance of the fleece. Later the gait becomes unsteady and after three or four weeks the sheep shows signs of pruritus. A detailed description of the terminal stages of the disease is given and it is stated that the disease usually lasts four to six months, occasionally a shorter time, rarely longer, and no form of therapy is known which significantly affects its duration.

The writers then state that most of their myopathic sheep conformed to this clinical syndrome, which they say is identical with classical descriptions of scrapie, but there were also several other clinically distinct syndromes in which myopathy also appeared to be the prime lesion. From their observations they have evolved a classification of the principal syndromes of myopathy in sheep, these are

	Cases seen	Post-mortem *examination in*
1. Classical scrapie	313	26
2. Non-wasting scrapie	6	2
3. Pining scrapie	28	6
4. Pre-clinical scrapie inapparent myopathy	5	5
5. Hind-limb palsy (closely related to scrapie)	14	4
6. *Post-partum* pining	73	2
	439	45

No reliable information is available about the general prevalence of this group of myopathic diseases and the sample of flocks considered in the paper should not, in the opinion of the authors, be considered representative of British sheep.

In the discussion it is pointed out that the discovery of muscle degeneration (myopathy) in sheep with the syndromes described in the paper goes far to explain the principal clinical signs found in these diseases. They emphasise that there is as yet no satisfactory explanation of the rubbing seen in many cases, nor of the curious changes in behaviour.

However, they claim, the discovery of myopathy as the only clear-cut pathological disorder in sheep with scrapie gives this disease for the first time a satisfactory pathological basis. They consider that the lesion in the muscles is a primary intrinsic degeneration of the muscle-fibres. There is no evidence that it is secondary to disease of the central nervous system, such as motor-neurone disease, or to peripheral neuritis, or that it is caused by vascular lesions such as polyarteritis. There follows a section drawing a comparison between these diseases in sheep, and human diseases. Consideration is then given to possible relation to other diseases such as stiff-lamb disease and muscular dystrophy in calves. The writers conclude that it is unlikely the myopathy they describe is a muscular dystrophy of the vitamin-E-controlled type. The possibility of myopathy in sheep being associated with hereditary factors is discussed briefly.

Finally, the problem of scrapie is reviewed in the light of the new discovery of myopathy in sheep and the discovery that complete necropsies of 34 consecutive cases of undoubted scrapie have all shown the myopathic lesions.

G. F. B.

News and Comment

B.V.A. AUTUMN CONFERENCE

With this issue is included a further copy of the Conference programme, bearing a tear-off slip on which members are asked to apply for admission tickets. Members are asked to send this to the General Secretary at B.V.A. Headquarters as soon as possible if they intend to be present.

It would be a great help if members would *definitely indicate* on the slip whether or not they require luncheon reservations made for them.

Tickets will not be sent immediately, but will be forwarded, nearer the time of the Conference, together with copies of all the papers.

THE ROYAL SOCIETY OF HEALTH

At a recent meeting of the Council of the Royal Society of Health, Professor Harold Burrow was installed as Chairman of Council for the year 1956-7. He succeeds Dr. Albert Parker, C.B.E. Professor Burrow is the first veterinary surgeon in the 80 years' history of the Society to become Chairman.

THE REGISTER FOR 1956

The attention of the Royal College of Veterinary Surgeons has been drawn to certain major errors in the 1956 issue of the Register. These concern the symbols for office holders, etc., under the list of abbreviations on page 13 and the references in the Index to pages mainly beyond page 450. The printers to the College have accepted responsibility for these errors and have undertaken to rectify them by sending to all members during the next few weeks an erratum slip for insertion in their copies.

ERRATA

In our issue of October 6th it was stated that Professor Wooldridge referred to the late Professor Shave as a former principal of the Royal Veterinary College. Unfortunately, he was incorrectly reported. Dr. Shave was for many years an eminent and most popular professor of anatomy at the Royal Veterinary College, but he was never principal of that college.

We are advised by Messrs. A. G. Nicholas & Co. that the name of their product mentioned in the first Abstract in last week's issue should have been spelt Megimide and not Negimide.

PROFESSOR J. S. S. INGLIS

As briefly announced last week, Mr. J. S. S. Inglis has just been elevated to the new Chair of Animal Husbandry and Veterinary Preventive Medicine in the University of Glasgow. The new professor was educated at Edinburgh University, the London School of Hygiene and Tropical Medicine, and qualified at the Royal (Dick) School of Veterinary Studies. He took the B.Sc. (Agric.) degree at Edinburgh.

The earlier part of his career was in Government service. He was on the research staff of the Northern Ireland Ministry of Agriculture from 1934 to 1936, and became a veterinary inspector for the Ministry of Agriculture from 1937 to 1943. In 1944 he was appointed lecturer at the " Dick " where he remained until he accepted an offer from the University of Glasgow where he has been senior lecturer in animal husbandry and preventive medicine until his new appointment.

Professor Inglis has a rich fund of experience in the field, in the laboratory, and as a teacher, and his elevation to a Chair places the seal on an already distinguished career.

DR. J. W. TREVAN

Our readers will have seen already in the daily press an announcement of the death of Dr. J. W. Trevan, F.R.C.P., F.R.S., at his home at Addiscombe, Surrey, on Saturday, October 13th, at the age of 69. He was a distinguished physiologist and pharmacologist and a pioneer in the application of statistical methods to the biological assay of drugs. He was pharmacologist to the Wellcome Physiological Research Laboratories from 1920 to 1940 and director of the Wellcome Research Laboratories at Beckenham, Kent, from 1940 to 1953.

His researches were concerned with bacterial toxins, snake venoms, the action of local anaesthetics, the physiology of the kidney, the nervous control of respiration, and traumatic shock.

In addition to his work for the Wellcome Foundation Dr. Trevan served on the Biological Standards Commission of the League of Nations, on the British Pharmacopoeia Commission, on several committees of the Medical and Agricultural Research Councils, and on the Board of Studies in Pharmacology of the University of London.

MAJOR J. H. WRIGHT, O.B.E., T.D., M.R.C.V.S.

Major J. H. Wright, O.B.E., T.D., of Linden Lea, Westwood Road, Heald Green, Cheadle, Cheshire, passed quietly away on October 10th, aged 83. He leaves two daughters and a son-in-law, to whom we extend our deepest sympathy. The funeral took place at Manchester Crematorium at 10.45 a.m. on Saturday, October 13th. Major Wright was a life member of the Lancashire Veterinary Association, having joined in 1906.

Mr. J. S. J. Lauder writes:—

With the death of John Henry Wright on October 10th the profession has lost one of the best-known veterinary surgeons in the north of England.

Major Wright who was 83, was born at Whitchurch, Shropshire, and received his professional training at the New Veterinary College, Edinburgh, where he qualified in 1894. After a short period with Cartwright's of Wolverhampton, he became assistant to Messrs. John and Alec Lawson, whom he ultimately succeeded.

He was one of the leading horse practitioners in the country, and as recently as 1954 he was the recipient of a presentation from the directors of the Manchester Racecourse Co., in recognition of his 50 years' service

as veterinary surgeon to the company. He acted in a similar capacity to the Haydock Park Racecourse.

During the 1914-18 war he served with distinction with the Lancashire Yeomanry, was awarded the O.B.E., mentioned in dispatches, and attained the rank of major. In recognition of his services with the Territorial Army he was awarded the T.D., an honour of which he was intensely proud.

As a dog show judge he was internationally known, having served in this capacity in France, Germany, Belgium, and America. He was equally well-known as an exhibitor of Fox terriers, Airedales, and Welsh terriers. He was a past member of the Kennel Club, Life President of the Welsh Terrier Association, former President of several other breed societies, and Chairman of the Manchester Championship Dog Society.

Among those present at the funeral, which took place on October 13th at the Manchester Crematorium, were the following members of the profession: T. S. Anderson, President of the Lancashire Veterinary Association, R. H. Oddy, J. W. Heney, and myself.

PERSONAL

Births

ARNOLD.—On September 22nd, 1956, to Prudence, wife of Roger Arnold, B.SC., M.R.C.V.S., of Mpwapwa, Tanganyika, a fourth daughter, Vivian Mary.

LIFTON.—On October 11th, 1956, to Wendy, wife of John M. Lifton, M.R.C.V.S., of Brunswick Road, Gloucester, a daughter, Melanie Jane.

NESTEL.—On September 18th, 1956, to Heather (M.R.C.V.S.), wife of Barry Nestel, B.SC., M.R.C.V.S., of Jamaica, British West Indies, a daughter, Penelope Susan, a sister for Carolyn.

Marriages

BICKNELL—CRAIG.—At University Chapel, Glasgow, on October 2nd, 1956, by the Rev. Thomas A. Balfour, M.A., Stephen Raymond, B.V.M.S., M.R.C.V.S., only son of Mr. and Mrs. Frederick Bicknell, Brondesbury, London, to Constance Rose, only daughter of Mr. and Mrs. Joseph Craig, Linburn Road, Glasgow.

LAWRENCE—KILBURN.—At St. Helen's Church, Sandal, Wakefield, on September 22nd, 1956, Brian Thomas Lawrence, M.R.C.V.S., of Finchley, N.2, to Heather Kilburn of Wakefield, Yorkshire.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

WRIGHT, John Henry, O.B.E., T.D., Linden Lea, Westwood Road, Heald Green, Cheadle, Cheshire, Major R.A.V.C. (T.A.) (Retd.). Graduated New Edinburgh, December 21st, 1894. Died October 10th, 1956, aged 83 years.

COMING EVENTS

October

24th (Wed.). Meeting of the R.A.V.C. Division, B.V.A., at the Zoological Society of London, Regent's Park, London, N.W.1, 12 noon.

26th (Fri.). Meeting of the Society for the Study of Animal Breeding at the Royal Veterinary College, Camden Town, N.W.1, 11.30 a.m.

Meeting of the Yorkshire Division, B.V.A., at the Royal Station Hotel, York, 2.30 p.m.

North of England Division, General Meeting, Agricultural Lecture Room, King's College, Newcastle upon Tyne, 2.30 p.m.

November

1st (Thurs.). General Meeting of the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 6 p.m.

7th, 8th and 9th (Wed., Thurs. and Fri.). B.V.A. Council and Committee Meetings in London.

15th (Thurs.). Autumn Meeting of the Eastern Counties Veterinary Society at the Suffolk Hotel, Bury St. Edmunds, 11.45 a.m.

16th (Fri.). Dinner-Dance of the Yorkshire Division, B.V.A., at the Royal Station Hotel, York.

22nd (Thurs.). Annual Dance of the Victoria Veterinary Benevolent Fund, County of Ayr Ladies' Guild at Western House, Ayr, 8 p.m.

22nd and 23rd (Thurs. and Fri.). B.V.A. Conference on Supplements and Additives in Animal Feedingstuffs at the Victoria Halls, Bloomsbury Square, London, W.C.1.

26th (Monday.). Annual Dinner of the Centaur Society at the Grand Hotel, Bristol, 7 p.m.

B.V.A. COMMITTEE AND COUNCIL MEETINGS IN LONDON

Wednesday, November 7th, at 7, Mansfield Street

10.00 a.m. Veterinary State Medicine Committee.
12.00 noon Parliamentary and Public Relations Committee.
2.15 p.m. Farm Live-stock Committee.
4.00 p.m. Home Appointments Committee.

Thursday, November 8th, at 7, Mansfield Street

10.00 a.m. Small-Animals Committee.
12.00 noon Finance Sub-committee.
2.00 p.m. General Purposes and Finance Committee.

Friday, November 9th, Connaught Rooms, Gt. Queen Street

10.30 a.m. Meeting of Council.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Cumberland. High Gate Close Farm, Troutbeck, Penrith (Oct. 11).

Hants. Bury Court Farm, Jenkyn Place Estate, Bentley, Farnham (Oct. 11).

Northants. Mansley Grange Farm, Broughton, Kettering (Oct. 10).

Staffs. Lightwood Lodge, Longton, Stoke-on-Trent; Kenroy, The Green, Cheadle (Oct. 11).

Fowl Pest

Essex. 256, Cressing Road, Braintree (Oct. 11).

Lancs. Plox Farm, Liverpool Road, Bretherton, Preston; The Woodlands, Crown Lane, Bartle (Oct. 9); 4, Plox Brow, Tarleton; School Farm, Cowhill, Grimsargh; 18, Blackgate Lane, Tarleton (Oct. 10); Stainfield House; Gill Lane, Longton; Windmill Cott, Liverpool Road, Bretherton; Grange Farm, Durton Lane, Broughton; Meadowside, Drumacre Lane, Longton, Preston (Oct. 11).

Swine Fever

Hants. Park Farm, Minstead, Lyndhurst (Oct. 10).

Lancs. Reeds Farm, Reeds Lane, Rainford, St. Helens (Oct. 10).

Norfolk. Manor Farm, Larling, Norwich (Oct. 10).

Somerset. Piggeries, Coombe Farm, Nunney, Frome; Wyke Farm, Wyke Champflower, Bruton (Oct. 11).

FOOT-AND-MOUTH DISEASE

The Foot-and-Mouth Disease Infected Area around Hambledon, Buckinghamshire, has been contracted as from October 18th to the following area:—

IN THE COUNTY OF BUCKINGHAM

The Borough of High Wycombe.

In the Petty Sessional Division of Wycombe (County): The parish of West Wycombe Rural.

In the Petty Sessional Division of Marlow: The parishes of Ibstone, Turville, Fingest and Lane End, Hambledon, Fawley, Medmenham, and Great Marlow.

IN THE COUNTY OF BERKS

In the Petty Sessional Division of Maidenhead: The parish of Remenham.

IN THE COUNTY OF OXFORD

The Borough of Henley-on-Thames.

In the Petty Sessional Division of Henley: The parishes of Pishill with Stonor, Bix, Nettlebed, and Rotherfield Greys.

In the Petty Sessional Division of Watlington: So much of the parishes of Watlington and Swyncombe as lies to the east of the road running from Shirburn via Watlington to Nettlebed including the said road.

The Foot-and-Mouth Disease Infected Area around Hambledon, Buckinghamshire, will be extended as from October 21st to include:—

IN THE COUNTY OF BUCKINGHAM

In the Petty Sessional Division of Wycombe (County): The parishes of Radnage and Stokenchurch.

IN THE COUNTY OF OXFORD

In the Petty Sessional Division of Watlington: So much of the parishes of Pyrton, Shirburn, and Lewknor as lies to the south and east of the road (B.4009) running from Watlington to Chinnor including the said road.

As from October 25th the whole of the Foot-and-Mouth Disease Infected Area around Hambledon, Buckinghamshire, will be released from restrictions.

The area described in the Schedule below will cease to be an Infected Area for the purpose of preventing the spread of foot-and-mouth disease on October 28th:—

IN THE COUNTY OF BUCKINGHAM

In the Petty Sessional Division of Aylesbury: So much of the parish of Haddenham as lies to the south and west of the railway running from Dorton to Princes Risborough excluding the said railway.

In the Petty Sessional Division of Brill: The parishes of Boarstall, Oakley, Worminghall, Ickford, Shabbington, Dorton, Chilton, Chearsley, and Long Crendon, and so much of the parishes of Brill, Wotton Underwood, and Ashendon as lies to the south and west of the road running from Ludgershall via Wooton Station and Upper Pollicott to Cuddington including the said road.

IN THE COUNTY OF OXFORD

In the Petty Sessional Division of Bullingdon: The parishes of Horton cum Studley, Stanton St. John, Thomley, Waterperry, Holton, Forest Hill with Shotover, Wheatley, Waterstock, Great Milton, Tiddington with Albury, Cuddesdon, Little Milton, Great Haseley, Thame, and Tetsworth.

Release from Restrictions of the Foot-and-Mouth Disease Infected Area around Rockley, Marlborough, Wilts.

The area described in the schedule below will cease to be an Infected Area for the purpose of preventing the spread of foot-and-mouth disease on October 23rd:—

IN THE COUNTY OF WILTS

The Borough of Swindon.

In the Petty Sessional Division of Cricklade: The parishes of Broad Town and Clyffe Pypard and so much of the parishes of Lydiard Millicent, Lydiard Tregoze, and Wootton Bassett as lies to the south and east of the road running from Purton via Wootton Bassett to Lyneham excluding the said road.

In the Petty Sessional Division of Marlborough: The parishes of Broad Hinton, Winterbourne Bassett, Berwick Bassett, and Winterbourne Monkton, so much of the parishes of Ogbourne St. George and Ogbourne St. Andrew as lies to the west of the main road (A.345) running from Chisledon to Marlborough including the said road, and so much of the parish of Preshute as lies to the north of the main road (A.4) running from Marlborough to Fyfield excluding the said road.

In the Petty Sessional Division of Swindon: The parishes of Wroughton and Chisledon and so much of the parish of Liddington as lies to the north and west of the road running from Wanborough via Liddington to Badbury including the said road.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Salmonellosis

Sir,—With reference to Mr. Tuffery's letter (*Vet. Rec.* **68,** 703) it would seem to me that the kernel of this particular nut lies in his last sentence: "For an epidemic to break out there must be a pathogenic micro-organism somewhere, but surely there need not always be a dietary deficiency?" It would be very difficult to say what organisms, normally inhabiting the gut of any animal are pathogenic or non-pathogenic. What really concerns us is why, in this particular brew, *Salmonellae* rise to the top? I do not think that anyone would dispute that if there were no *Salmonellae* there would be no epidemic salmonellosis. On the other hand, it is my experience, if, basically, nutrition is at fault and *Salmonellae* are absent, then other microbic diseases replace salmonellosis and the net loss, mortality and morbidity, is the same.

As for dietary deficiencies; in this "atomic age" progress of nutrition, new essential factors for some or other animal are being constantly unearthed. I would hesitate very much to discuss deficiencies in this connexion, nor have I ever suggested that dietary deficiencies have any bearing on epidemic salmonellosis.

What I do suggest, and recommend for serious consideration, is that dietary *stress* factors favour nutrition of the microbe more than the nutrition of the animal. These stress factors are now beginning to receive attention and I will enumerate a few. (a) The residual oils of *processed* proteins, oil seeds, fish, meat, and other animal products. These appear to act via oxidation of vitamins A and E. (b) The denatured moiety of processed proteins in which the breaking of the chain or side chain linkage of amino-acids prevent the utilisation of the amino-acid by the animal but afford excellent food for bacteria. (The prime function of bacteria is to break complicated organic compounds down to simple elements.) (c) Secondary mechanical substances such as porous fibre, *e.g.* bran, which afford a nidus for the proliferation of certain organisms, *e.g.* anaerobes or micro-aerophiles. The feeding of protein beyond the immediate requirement of the animal thus leaving residues for bacteria to thrive upon. The continuous feeding of an unvaried diet which tends to stabilise the bacterial flora of the gut, with some species adopting a dominant rôle.

Interwoven, is the understanding of the true function of bacteria. If we set up the manure heap within the animal we will grow the organisms which inhabit the manure heap. Our knowledge of the effects of symbiosis and the synergistic action of one bacterium upon another is almost nil, but it is certain, given the right conditions of nutrition, that *Salmonellae* will rise to the top of the brew. Thereafter, it is only a question of virulence of the organism by passage or the immunological properties of the host which determines the nature of the epidemic.

My experience is, that with the maximum of nutrition to nourish the host, coupled with the minimum of nutrition to nourish the germ, I escape epidemic salmonellosis.

Yours faithfully,

T. BARTON MANN.

" Braust,"
Weyhill,
nr. Andover, Hants.
October 6th, 1956.

The Husbandry and Diseases of Pigs

Sir,—I should like to endorse Mr. Hogg's remarks (*Vet. Rec.*, **68**, 704) on a necrosis of the foot-pad in pigs resembling " foot-rot." I, also, looked for some reference to it in the B.V.A.'s excellent revised publication, hoping to find some information on its pathology.

The late Professor Macqueen's wise dictum regarding horses—if they are lame in the head, examine the foot—applies equally to pigs. I find surprising the number of adult pigs, especially sows, showing symptoms suggestive of laminitis or rheumatism which, when examined, are found to be suffering from the condition referred to.

In the past, I have found the condition most resistant to treatment. Messrs. Parke Davis & Co., fairly recently, kindly supplied me with some chloromycetin tincture 10 per cent. as used for foot-rot in sheep. In the limited trial I have been able to give it, the results have been spectacular.

Yours faithfully,

" Braidwood,"
Acle,
Nr. Norwich.
October 12th, 1956.

O. V. GUNNING.

Monozygous Twins

Sir,—Mr. G. H. Arthur's review on the incidence of monozygous twins published in your issue of September 22nd relies throughout on the evidence of finding a single corpus luteum but two foetuses.

One cannot help but wonder how often the phenomenon seen in the attached photograph occurs where

two ova are found within a single follicle which presumably results also in the formation of a single corpus luteum.

Yours faithfully,

L. E. ROWSON,
Director.

Cambridge and District Cattle Breeders Limited,
The Gravel Pits,
Huntingdon Road,
Cambridge.
October 6th, 1956.

The fortnightly return under the Diseases of Animals Act, 1950, will be published next week.